Environmental Pollution, Human Malignancy, and Public Policy

Ronald A. Harris and George M. Lane

Southern University

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Abstract

This is a spatial and temporal study of environmental pollution, human malignancy, and public policy in Louisiana, from 1988 to 2002. We focus research on benzene exposure and the incidence of Acute Myelogenous Leukemia (AML). Benzene is a carcinogenic chemical, gasoline additive, and industrial solvent. When exposed to benzene, humans develop AML. Louisiana Tumor Registry (LTR) data on AML are linked to the Toxic Release Inventory (TRI) data on environmental benzene releases to estimate statistical models and map spatial relationships. We determine that the mean number of benzene reporting facilities best explains human malignancy rates (AML) in Louisiana. White males are more often affected by AML than blacks and females, which contradict the environmental racism thesis that environmental pollution targets poor blacks. Rather, white males are likely affected at work by benzene exposure in petrochemical plants.

Cancer Alley Redux

Social justice is the pursuit of environmental activists who write about environmental racism and class exploitation in the South (see Bullard, 1990). Florence Robinson said, “The areas in Louisiana that have the highest toxic-release discharges are well over 50 percent African-American" (Little, 2005). She refers to the 85 mile stretch from Baton Rouge to New Orleans as “Cancer Alley,” because more than 120 chemical plants and petroleum processors are located along this part of the Mississippi River. Says Robinson, “the greater the percentage of minority residents, the greater the pollution” (Gupta, 2005).

At first glance it appears that blacks may be targeted by petrochemical polluters, except that blacks are not a minority along Cancer Alley. While blacks are 12% of the U.S. population, they comprise 33% of Louisiana’s population, (McKinnon, 2001). In Baton Rouge, blacks are 50% of the population and in New Orleans blacks are 67% of

the population (US Census Bureau, 2006). Environmental justice advocates deal in the doors of perception—deriving evidence from surveys of social attitudes about pollution, rather than facts. We studied environmental pollution and policymaking in Louisiana, seeking to clarify and verify the thesis of environmental justice and racism, as alleged.

We began by seeking to specify a crucial experiment to test the environmental justice or racism thesis, which argues that race and class may explain cancer incidence along the Mississippi corridor, from Baton Rouge to New Orleans in South Louisiana. Known as "cancer alley," this segment of geography along the river has a concentration of petrochemical facilities that emit pollutants that potentially cause human malignancy. Because human malignancy is associated with many environmental and lifestyle factors, we chose a proven human hazard that could test the environmental justice hypothesis. We focused on reported benzene releases and acute myelogenous leukemia incidence.

Environmental Pollution

Benzene is a chemical carcinogen linked to Acute Myelogenous Leukemia (AML). This type of leukemia is also caused by environmental radiation exposures, like inappropriate radiological shielding, therapeutic irradiation, and atomic bomb explosions. AML rates are higher among blacks, males, and the elderly. Louisiana is ranked second after Texas in benzene releases to the environment. These two states contain the most petrochemical plants in the nation. Firms that produce benzene have been shown to violate regulatory prohibitions and could harm human health.

Properties and uses of Benzene

Benzene is a member of aromatic hydrocarbons recovered from refinery streams. Its one of the world's major commodity chemicals. 85% of benzene produced is used to produce

other chemicals, like styrene cumene and cyclohexane. Benzene is raw material used in synthetic rubbers, gums, lubricants, dyes, pharmaceuticals, and agricultural chemicals. The ban of tetraethyl lead in gasoline has led to an increase in the aromatic hydrocarbon content of gasoline to maintain high-octane levels and antiknock properties. Gasoline contains less than 2% benzene by volume in the United States of America, but in many foreign countries, benzene concentration in gasoline may be as high as 5% by volume.

Because is an excellent solvent. Its use in paints, thinners, inks, adhesives, and rubbers, however, is decreasing and accounts for less than 2% of benzene production. Benzene was replaced mostly by toluene or chlorinated solvents. Although benzene is no longer added in significant quantities to most commercial products, traces of it may still be present as a contaminant. Benzene is now widespread in the environment. Airborne benzene is typically produced by the chemical manufacturing or the gasoline industry, including gasoline storage facilities and combustion engines, such as automobiles and lawn mowers. Thus, benzene is a component of both indoor and outdoor air pollution.

Industrial use and exposure to benzene

Workers in industries using or producing benzene such as petrochemical companies, petroleum refining and coke and coal chemical manufacturing, rubber tire manufacturing, and companies involved in the storage or transport of benzene and petroleum products containing benzene, have the greatest potential to be exposed to benzene (see Austin and Cole, 1988). The Occupational Safety and Health Administration (OSHA) estimates that 238,000 workers in the United States may be exposed to benzene in refining operations, gasoline production, chemical manufacturing, and plastics and rubber manufacturing. In

other occupations, like steel workers, printers, rubber workers, shoe makers, laboratory technicians, and gas station employees, workers may be similarly exposed to benzene.

Atmospheric benzene levels of up to 6.6 ppm and 6-hour TWAs (time-weighted averages) of 0.1 ppm have been measured during gasoline pumping. This risk has been lowered by installing vapor recapture devices on delivery hoses. These devices, if used properly, significantly reduce exposure. Catalytic converters have significantly reduced the benzene in automobile emissions (see Federal Register, 1987). Workers employed in industries using or producing benzene have a higher chance of exposure (see Proctor, 1997; Patty's Industrial Hygiene and Toxicology, 1978; Westbury v. Gislaved, 2001).

Health effects of benzene exposure

Benzene is rapidly and extensively absorbed by inhalation and ingestion. In humans, approximately 50% of inhaled benzene is absorbed after a 4-hour exposure to about 50 ppm benzene in air (see Hallenbeck and Flowers, 1992). After an exposure, benzene is found throughout the body, but it collects into the bone marrow and tissues with high lipid, or fat content. Thus, autopsies of people who died after acute exposure showed that lipid-rich tissues, such as the brain and fat, and tissues, such as the kidney and the liver, have higher levels of benzene than other tissues. Once absorbed, benzene is metabolized in the liver and later in the bone marrow (see Paxton et al., 1994).

Benzene can cause dangerous hematologic toxicity such as anemia, leukopenia, thrombocytopenia, or pancytopenia after chronic exposure. These effects are believed to be caused by the metabolites of benzene, which damage the DNA of the "pluripotential" stem cells. Pluripotential hemopoietic stem cells or pluripotential hematopoietic stem cells (PHSCs) are stem cells found in the bone marrow. PHSC are the precursor cells

which give rise to all the blood cell types of both myeloid and lymphoid lineages. All of the blood's components may be affected to varying degrees (see Rinsky et al., 1987). The accelerated destruction or reduction in the number of all three major types of blood cells is termed pancytopenia. Fatal infections can also develop if granulocytopenia is present, and hemorrhage can occur as a result of thrombocytopenia (see Collins et al., 1991).

Human Malignancy

Human malignancy refers to health problems that include cancers and related illnesses. We focus on acute myelogenous leukemia (AML) and its relation to benzene exposure.

Acute Myelogenous Leukemia

Acute myelogenous leukemia (AML) is a cancer of the blood system in which there is an abnormal production of hematologic stem cells, granulocytic leukocytes, red blood cells and platelets (see Ringen and Addis, 1983; Holmberg and Lundberg, 1985; Kalnas and Teitelbaum, 2000). AML is mostly observed in adults and increases with age. AML variants include promyelocytic leukemia, and erythroleukemia (see Korte et al, 2000; Rinsky et al., 1987; NIOSH Standards, 1976; Wong, 1987; Yin et al., 1996).

The medical literature discloses cases of AML where benzene exposure is the primary agent (see Cairns, 1979; WHO International Agency for Research on Cancer, 1987; American Petroleum Institute, 1948). The relatively common description of aplastic anemia associated with benzene exposure followed through a pre-leukemic phase into acute leukemia further supports the theory that the bone marrow toxicity of benzene is connected to diseases such as anemia, thrombocytopenia, leukemia, or the other hematological diseases (see Infante and Book, 1992; Picciano, 1980; Hayes, et al., 1997).

Cigarette smoking associated with an increased risk of leukemia

Cigarette smoking increases the risk for leukemia. Benzene, an established leukemogen, is present in cigarette smoke (see Goldstein, 1988. Persons who smoke have a small but statistically significant increased risk of developing AML. Researchers have determined the proportion of total leukemia and partial AML attributable to the benzene in cigarette smoke. Lifetime risks of total leukemia and AML deaths for non-smokers and light and heavy smokers can be calculated with life tables. The benzene in cigarette smoke is estimated to cause from one-tenth to one-half of smoking-induced total leukemia mortality and up to three-fifths of smoking-related AML mortality. However, we have not measured smoking rates in Louisiana and assume smoking is randomly distributed.

Public Policies

There are myriad policies promulgated by government seeking to address the causes and consequences of environmental pollution and human malignancy. In sections below, we discuss standards for air, water, and food. These regulations are summarized in Table 1 (Appendix).

In 1987, the US Occupational Safety Health Administration (OSHA) instituted a PEL for benzene of 1 ppm, measured as an 8-hour TWA (time-weighted average), and a short-term exposure limit of 5 ppm. These legal limits were based on studies showing health risk to workers exposed to benzene. The risk from exposure to 1 ppm for a working lifetime has been estimated as 5 excess leukemia deaths per 1,000 employees exposed. No threshold was assumed for benzene's carcinogenic effects. OSHA also established an action level of 0.5 ppm to encourage even lower workplace exposures. The National Institute for Occupational Safety and Health (NIOSH) recommends an exposure limit of 0.1 ppm as a 10-hour TWA. NIOSH recommends that benzene be handled in the

workplace as a human carcinogen. In 1997, the American Conference of Governmental Industrial hygienists lowered its TWA-threshold limit value to 0.5 ppm to reflect the regulatory change in cancer classification to A1 (i.e., confirmed human carcinogen).

Under section 112 of the Clean Air Act, benzene is classified as a hazardous air pollutant. EPA has not set a specific ambient air standard for benzene but has imposed restrictions designed to lower industrial emissions of benzene by 90% over the next 20 years. In addition, regulations have been proposed that would control benzene emissions from industrial solvent use, waste operations, transfer operations, and gasoline marketing.

At gas stations, proposed rules would require new equipment restricting benzene emissions while storage tanks are being filled. Under the Clean Air Act Amendments of 1990, the use of clean ("oxygenated") fuels was mandated as a means of reducing motor vehicle emission-related air pollutants. EPA predicts that this clean fuels program will decrease ambient benzene levels by 33%.

The National Primary Drinking Water Regulations promulgated by EPA in 1987 set a maximum contaminant level for benzene of 0.005 ppm (5 ppb). This regulation is based on preventing benzene leukemogenesis. The maximum contaminant level goal, a non-enforceable health goal that would allow an adequate margin of safety for the prevention of adverse effects, is zero benzene concentration in drinking water.

Benzene is a precursor in soft drinks! Effective April 1988, the US Food and Drug Administration (FDA) mandated that benzene can only be an indirect food additive in adhesives used for food packaging. A dispute has erupted between the FDA and the Environmental Working Group (EWG) over whether some bottled and canned beverages contain unhealthy levels of benzene, a known carcinogen. Some drinks contain ascorbic

acid (vitamin C) and the preservative sodium benzoate. In the presence of ascorbic acid under certain conditions, benzene is formed through the decarboxylation of benzoic acid (see Brackett, 2006). FDA first became aware of the problem in 1990 and asked manufacturers to reformulate their beverages.

Theory

The environmental racism hypotheses posits that poor blacks are exposed more often to harmful industrial pollutants in the environment than rich whites who can relocate by voting with their feet, so to speak. This is a "not-in-my-backyard" NIMBY problem.

Benzene releases to the environment may be malicious, deliberate, incidental or accidental. Neoclassical economic theory predicts that the costs of internalizing third-party effects from petrochemical manufacture should reduce profits at the margin. The negative external byproducts of petrochemical production include toxins that pollute the environment which harm human health. Third-party costs of externalities in the form of environmental degradation constitute a market failure. Market failure is a conventional rationale for government intervention in the marketplace with corrective instruments, such as public policies. Aside from the collection action problem of inducing firms to correct polluting behavior through selective incentives, such as regulations, we should expect environmental pollution effects to be equally distributed among all members of society, in the lack of other evidence.

Derivation of Hypotheses

We derive general hypotheses (1) from the theory of environmental racism and (2) based on evidence of an occupational health risk for benzene exposure at petrochemical plants.

H_1: Humans near benzene releases have higher AML rates than otherwise.

H_2: Blacks have higher rates of acute myelogenous leukemia than whites.

H_3: Males have higher rates of acute myelogenous leukemia than females.

These hypotheses were tested with statistical models using empirical data. The results of estimation are discussed in sections below. There are limits to this study because of data.

Limitations of the Study

Despite obtaining approval for research from our IRB for human subjects on campus at Southern University, Louisiana's Tumor Registry would not release point data or counts of less than six (6) cases. This rule is designed to protect the confidentiality of human subjects, among other things. Therefore, we are limited to rates and counts and have no point data to show or analyze for spatial relationships. Further, these data cover all ages, including adults and children. So we cannot measure the effect of age on getting AML.

Not all benzene releases into the environment can be calculated, measured, or captured. For example, there is mobile benzene in gasoline powered vehicles, such as cars and trucks. To proxy mobile benzene, the human population captures it implicitly. Industry has an essential role to play in the effort to clean up our air and water, but it cannot be expected to reliably police itself. While section 372.18 of the TRI legislation states, "Violators of the requirements of this part shall be liable for a civil penalty in an amount not to exceed $25,000 each day for each violation as provided in section 325(c) of Title III", no provision is provided mandating the accuracy of data submitted. Until an impartial reporting system is created by environmental regulators rather than chemical

manufacturers, relying on a potential polluter to accurately assess its performance will continue to produce unreliable data and uncertain protection for Louisiana's residents.

Measurement

We looked at benzene in every parish in Louisiana that reported releases to the Toxic Release Inventory (TRI) and for Acute Myelogenous Leukemia rates, from 1988-2002. Data on the incidence of AML were obtained from Louisiana's Tumor Registry (LTR). LTR provided data on rates and counts from 1988 to 2002 for this study. These data are age-adjusted to population movements and demographics for the 2000 census. The US EPA's Toxic Release Inventory (TRI) data for facilities reporting benzene were obtained from The Right-to-Know Network (RTK), a public service provided via OMB Watch. TRI is a publicly available database that contains information on toxic chemical releases. This inventory was established under the Emergency Planning and Community Right-to-Know Act of 1986 (EPCRA) and expanded by the Pollution Prevention Act of 1990.

Reliability and Validity

Individual facilities send TRI data in reports to the EPA every year. On RTK NET, users can search the data by geographic area, facility, industry, parent company, and offsite waste transfer data, to learn which toxic chemicals are present, and in what amounts. The EPA is currently pursuing plans to reduce the amount of data collected on toxic pollution throughout the country, which would severely damage the usefulness of the TRI program.

EPA is checking to see that the same facility keeps the same TRI Facility ID from one year to the next, and that the cities, states, counties, and Zip codes of facilities match each other. However, the locations and ID numbers of off-site transfer destinations and POTWs (Publicly Owned Treatment Works) are not checked. EPA queries facilities that

send submissions with very high release numbers, and facilities whose numbers change drastically from one year to the next, to see if the numbers are correct. There is also a program under which TRI facilities can be inspected (not many are). Other than this, the numbers that a facility submits are not checked. Facilities that report can estimate their numbers. They are not required to be measured. Pollution releases reported to TRI are generally assumed to be legal, under the assumption that a facility would be foolish to report illegal releases rather than falsifying their TRI reports.

There are special problems with the waste generation numbers reported in Section 8 of the Form R. Because of interference by the Office of Management and Budget, EPA was never able to publish a definition of important concepts involved in this part of the form. As a result, the distinction between recycling, which is reportable to TRI, and reuse within a process, which is not, is unclear, and different facilities probably have different standards for reporting these quantities. TRI quantities are supposed to be estimated to only two significant digits. That means that if you add up a number of TRI releases and get a number like 11,264,586 pounds; only the first two digits have any meaning. In most cases, you would be better off writing this number as 11 million pounds by rounding off.

Therefore, not all, or even most, pollution is reported in TRI. However, TRI does have certain advantages. It is multi-media. Facilities must report the amounts they release to air, land, water, and underground separately, and must report how much they send off-site. All quantities are reported as amounts of toxic chemical (in pounds for all chemicals except Dioxin, which is in grams). This is an advantage over other databases that report releases as concentrations, or which report releases by volume of waste. These measures are often impossible to convert into pounds. Here we have a standardized volume metric.

Variables

Acute Myelogenous Leukemia. The data were operationalized as variables for statistical modeling. AML is the dependent variable, which was measured as the age-adjusted rate per 100,000 population. These data are available for all races, male and female, together with break-outs for gender (male, female) and race (black, white, and other). These data are only available at the parish level, a condition of release imposed by LTR. Further, the LTR data are truncated such that for less than six cases in any parish, either (1) no data were reported or (2) no calculations of rates per 100,000 were made. Thus, the unit of analysis is the parish. Table 2 shows the parishes that have benzene reporting facilities (Appendix).

Figure 1 shows that AML rates have increased in Louisiana, from 1988 to 2002. This is an inverse relationship with benzene releases (see Figure 2 below), and contrary to expectations. There may a time delay in measuring harm to human health from onset of chemical exposure in the environment. We may see now the effects of past releases.

Benzene Releases. These data were extracted from the Right-to-Know Network. The facilities releasing benzene must meet a minimum threshold required for reporting. Therefore, not all of the benzene releases by parish will appear in EPA's TRI database.

The Toxic Release Inventory (TRI) is a database of information about releases and transfers of toxic chemicals from facilities in certain industrial sectors, including manufacturing, waste handling, mining, and electricity generation. Facilities must also report the total amount of toxic chemicals in waste that they produce. Facilities must report to TRI if they fulfill four criteria: They must be a manufacturing facility (primary SIC code in 20-39) or in one of a number of non-manufacturing industries added for the

1998 reporting year; they must have the equivalent of 10 full- time workers; they must either manufacture or process more than 25,000 lbs of the chemical or use more than 10,000 lbs during the year; and the chemical must be on the TRI list of over 600 specific toxic chemicals or chemical categories. Importantly, benzene releases come from many different sources, both fixed and mobile (e.g., from automobiles burning fossil fuels).

Benzene releases are reported as (1) fugitive air, (2) stack air, (3) water, (4) on-site transfers, and (5) off-site transfers. We are less concerned here about water releases of benzene into the environment, as they are indirect exposures. Air releases are direct. Figure 2 indicates that benzene releases in Louisiana have decreased, from 1988-2002. While this suggests that environmental regulation is working to reduce harmful chemical exposures, there is reason to believe that chemical releases often go reported. The trend line spike for stack air releases of benzene in 1997 is not a data artifact! Rather, the TRI reporting system yields wide variances in pollution estimates (Houston Chronicle, 2006).

Fugitive Air Releases of Benzene. Fugitive air emissions are all releases to air that are not released through a confined air stream. Fugitive emissions include equipment leaks, evaporative losses from surface impoundments and spills, and releases from building ventilation systems.

Stack Air Releases of Benzene. Stack or point source air emissions occur through confined air streams such as stack, vents, ducts, or pipes.

On-Site Transfers of Benzene. An on-site transfer of a TRI chemical is one that may transfer the chemical to a specific on-site disposal or recycling inside the facility.

Off-Site Transfers of Benzene. A transfer of a TRI chemical off-site in waste from a TRI facility to another destination. Transfers are divided into publicly owned treatment

works, or sewerage plants and other off-site transfers. The other off-site transfers include recycling, energy recovery (burning the chemical as fuel), treatment, or disposal. EPA treats transfers to disposal as "off-site releases". RTK does not. If a TRI chemical is sent off-site as part of a product, this is not reported within TRI. The data for water releases of benzene were collected, but not included in statistical models, because they are indirect.

Methodology

Two modeling approaches were used to analyze the data for benzene releases and cancer rates in Louisiana. First, statistical models were used to estimate spatial and temporal relationships. Second, maps were used to visually display information about these data.

Statistical Models

Statistical models using t-tests of sample means and linear regression were estimated to determine the relationships of explanatory variables for AML rates among Louisiana's sixty-four parishes, from 1988 to 2002. The results of estimation are discussed below.

Independent Samples T-tests. We compare AML rates for all races and genders, by race and gender, between Louisiana's parishes with and without facilities that report benzene releases. Only twenty-one about one-third (21/64) of Louisiana's parishes have facilities required to report benzene releases. Facilities in the other forty-three parishes were not required to report benzene releases under the Toxic Release Inventory. Because of data truncation by LTR, valid AML rates are available for only 87.5% of the parishes.

An independent samples t-test was conducted to compare the mean difference on AML rates for all races, genders, between parishes with and without benzene reporting facilities. The mean AML rate for parishes with benzene reporting facilities is 3.105 (n=20), while the mean AML rate for parishes without benzene reporting facilities is

3.572 (n=36). The difference of mean AML rates between parishes is 0.462. Parishes without benzene reporting facilities have higher mean AML rates for all races, genders, than parishes with benzene reporting facilities. This difference is statistically significant (p=0.050), when variances are assumed unequal under Levene's test. When the sample variances are assumed equal, no statistically significant difference was found (p=0.082).

The only other result, using an independent samples t-test, was found for average AML rates among black females. The mean AML rate among black females for parishes with benzene reporting facilities is 1.880 (n=10), while the mean AML rate among black females for parishes without benzene reporting facilities is 0.343 (n=14). The difference of means is 1.537. As expected, parishes with benzene reporting facilities have higher AML rates for black females on average than those parishes without benzene reporting facilities. This difference of means is statistically significant: regardless of whether the variances are assumed either equal (p=0.010) or unequal (p=0.025) using Levene's test.

Linear regression. We predicted AML rates for all races and genders across parishes from 1988-2002 using linear regression models. Because of correlation among the regressors, the models were specified to manage multicollinearity. Mean number of benzene reporting facilities is positively and significantly correlated with total benzene releases (r=0.846, p=0.000, n=21), as expected. Stack air releases, fugitive air releases, water releases, on-site transfers, and off-site transfers together: comprise total benzene releases and are highly correlated with each other and with benzene reporting facilities.

Only the mean number of reporting facilities or total benzene releases could be used for the independent regressors. The regressand was either AML for all races and genders or subsets of AML rates by race and/ or gender. A pooled time series analysis

was not conducted, because the matrix cells were zero or truncated for many parishes in particular years. Hence, only bivariate regression models of AML rates were estimated. Table 3 (Appendix) shows the results of estimation for AML rates (all races and genders) predicted by mean number of reporting facilities. Goodness-of-fit statistics indicate that the model explains one-fifth of the variance (Adj. R^2=.211) and is statistically significant (.024).

The results for whites only and white males is consistent with the results for all races and genders (see Tables 4 and 5, Appendix). The revelation that (1) all races and genders are affected or that (2) whites are affected or that (3) white males are affected environmental exposure to benzene, runs contrary to expectations for blacks in general and black males in particular being most at risk for AML. Surprisingly, no statistically significant results were found for bivariate estimates of AML rates when using total benzene released. The implication is that proximity to petrochemical plants rather than benzene releases is more explanatory of AML rates. It suggests that point data for AML cases should be useful.

Spatial models. Spatial modeling was conducted with a Geographic Information System (GIS) constructed in ArcView 9.1 to map Louisiana's sixty-four parishes. A map shows AML rates by parish for the general population using a violet color ramp. The benzene reporting facilities are shown as black diamond markers (NFPA Chemical 6). Total releases of benzene is shown as yellow circles (NFPA Chemical) that grow in size in relation to the quantity. While the concentration of benzene reporting facilities shows close correspondence with total benzene released, the incidence of AML does not relate.

Results

We have statistical estimates that at first glance may appear contradictory. Using t-tests, we find that the mean AML rates for all races and genders is higher for parishes with no benzene reporting facilities. This is contrary to expectations and suggests that AML rates are not related to benzene reporting facilities. But we also find that mean AML rates are higher among black females, as expected. However, there are fewer observations for the black female population, due to truncation of the LTR database for AML. Therefore, the hypothesis "humans near benzene releases have higher AML rates than otherwise" is not supported, except perhaps for black females. That would be evidence for environmental racism, except that these data are parish-wide at present and point data would be better.

Among bivariate regressions, we find that the AML rate for all races and genders increases as the average number of benzene reporting facilities increases among parishes having facilities. We do not find support for the hypothesis "blacks have higher rates of acute myelogenous Leukemia than whites" for those parishes having benzene reporting facilities. Rather, we see that the general population, whites in general, and white males in particular, have AML rates than increase positively in relation to number of facilities.

Given limitations stemming from the data on Acute Myelogenous Leukemia rates, we cautiously make inferences. There are also limitations on measuring benzene releases to the environment, given reported variances. The hypothesis that "males have higher rates of acute myelogenous Leukemia than females" is contradicted in part and partly supported. T-test results reveal black females have higher AML rates when residing in parishes with benzene reporting facilities, while bivariate regression results show that white males have AML rates that increase in relation to increased number of facilities.

Conclusions

Protecting workers and the public from toxic chemicals and particularly carcinogens, has been a major goal of US public policy. In the absence of knowing by what mechanism of action a toxicant harms people, regulatory toxicology assumes that even tiny doses can cause harm. Risk has led to legislation and regulation that seek to ban toxic chemicals or lower exposure to trivial levels. Policy alternatives are discussed as recommendations.

Implications for Public Policy

Louisiana's racial demographics call into question the assertion that blacks are exposed to benzene releases more than are whites. However, the Environmental Protection Agency supports grassroots community movements under their environmental justice program.

Our findings suggest that black females may be affected by benzene as revealed through AML rates. White males are likewise affected. Black female AML rates need to be reinterpreted with point data to measure the distance between residences and benzene reporting facilities. White male AML rates should be supplemented with occupational data, as it probably explains why that population subgroup shows positive increases with facilities. These are preliminary results for a test of the environmental racism hypothesis, which appears to have received no additional support. Still further research is needed.

Policy Recommendations

Scientific advisory boards report health benefits from low-level exposure to toxicants, including some carcinogens. This is known as "hormesis". Hormesis is a fundamental change in the approach to regulating toxic substances. In particular, all toxicants that benefit health at low-level exposures would face similar change in regulations for low-dose exposure. The result would be the dissolving of differences for regulatory policy

covering chemical carcinogens and non-carcinogens at low doses. Two questions may require an answer before hormesis can be incorporated into current regulatory policy:

1. Are there sensitive individuals who would be harmed at doses that would help most people?

2. Is the hormetic effect toxicant specific or would exposure to just a few toxicants achieve the full benefit from hormesis?

Specifically, the available knowledge on the toxicity of benzene and the failure to take precautions to protect workers and the public in light of this knowledge over the past century has been and remains cause for concern. Inadequate actions by organizations and governments alike throw into question the ability of these organizations to protect the health of the public. In the case of benzene exposure in the workplace, the precautionary principle has not been relevant. Recommendations made in the 1920s for the substitution of benzene with other solvents known to be less toxic to bone marrow went unheeded for decades even though high percentages of workers being surveyed demonstrated blood disorders. Furthermore, benzene was not withdrawn from consumer products in the US until 1978. This was done voluntarily by manufacturers and it has not been validated.

Placing a warning label on gasoline pumps that includes the cancers and other diseases known, or likely to be caused by benzene exposure, may reduce unnecessary benzene exposure to garage mechanics, highway maintenance workers, and consumers who fill their own gas tanks, but who more unknowingly use the gasoline in consumer products at home, and use gasoline as a solvent without knowledge of its cancer risks.

Direction of Future Research

Future research will be conducted using point data (i.e. x,y coordinates), assuming that permission is obtained from the Louisiana Tumor Registry (2006). The demographics of

Louisiana call into question the proposition that blacks are exposed to benzene releases and suffer acute myelogenous leukemia more than whites. A detailed analysis should allow us to pursue this line of research when better quality data are obtained from LTR. With point data, we should be able to conduct contextual analyses over time and space.

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Appendix

Table 1. Summary of Current Standards and Regulations for Benzene

Agency	Focus	Level	Comment
American Conference of Governmental Industrial Hygienists	Air (workplace)	0.5 ppm	Advisory; TWA (Time weighted average); confirmed human carcinogen
American Conference of Governmental Industrial Hygienists	Air (workplace)	2.5 ppm	STEL (Short-term exposure limit 15-minute ceiling limit)
National Institute for Occupational Safety and Health	Air (workplace)	0.1 ppm	Advisory; 10-hour TWA
National Institute for Occupational Safety and Health	Air (workplace)	1.0 ppm	15-minute ceiling limit
Occupational Safety and Health Administration	Air (workplace)	1.0 ppm	Regulation; TWA
Occupational Safety and Health Administration	Air (workplace)	5.0 ppm	15-minute STEL
Occupational Safety and Health Administration	Air (workplace)	0.5 ppm	Action level TWA
USEPA	Water (drinking)	5 ppb	Maximum Contaminant Level
Food and Drug Administration	Food	N/A	May be used only as a component of packaging Adhesives

Table 2. Louisiana Parishes with Benzene Reporting Facilities

Parish	Average Number of Facilities Reporting
East Baton Rouge	7.53
Calcasieu	7.47
St. Charles	4.27
Ascension	3.40
Iberville	2.80
St. John the Baptist	2.80
St. James	2.40
St. Bernard	2.00
Plaquemines	1.33
Caddo	1.27
Assumption	1.15
Acadia	1.11
Claiborne	1.00
Jefferson	1.00
Jefferson Davis	1.00
Lafayette	1.00
La Salle	1.00
St. Landry	1.00
Terrebonne	1.00
Webster	1.00
West Baton Rouge	1.00

Table 3. Mean Benzene Reporting Facilities regressed on AML Rate for all cases

Variable	B	Standard Error	Beta	T	Significance
Constant	2.738	.200		13.669	.000
Facilities Mean	.166	.067	.502	2.466	.024

N=20, Adjusted R^2=.211, Sig. F=.024.

Table 4. Mean Benzene Reporting Facilities regressed on AML Rate for all whites

Variable	B	Standard Error	Beta	T	Significance
Constant	.2887	.201		14.348	.000
Facilities Mean	.170	.066	.529	2.572	.020

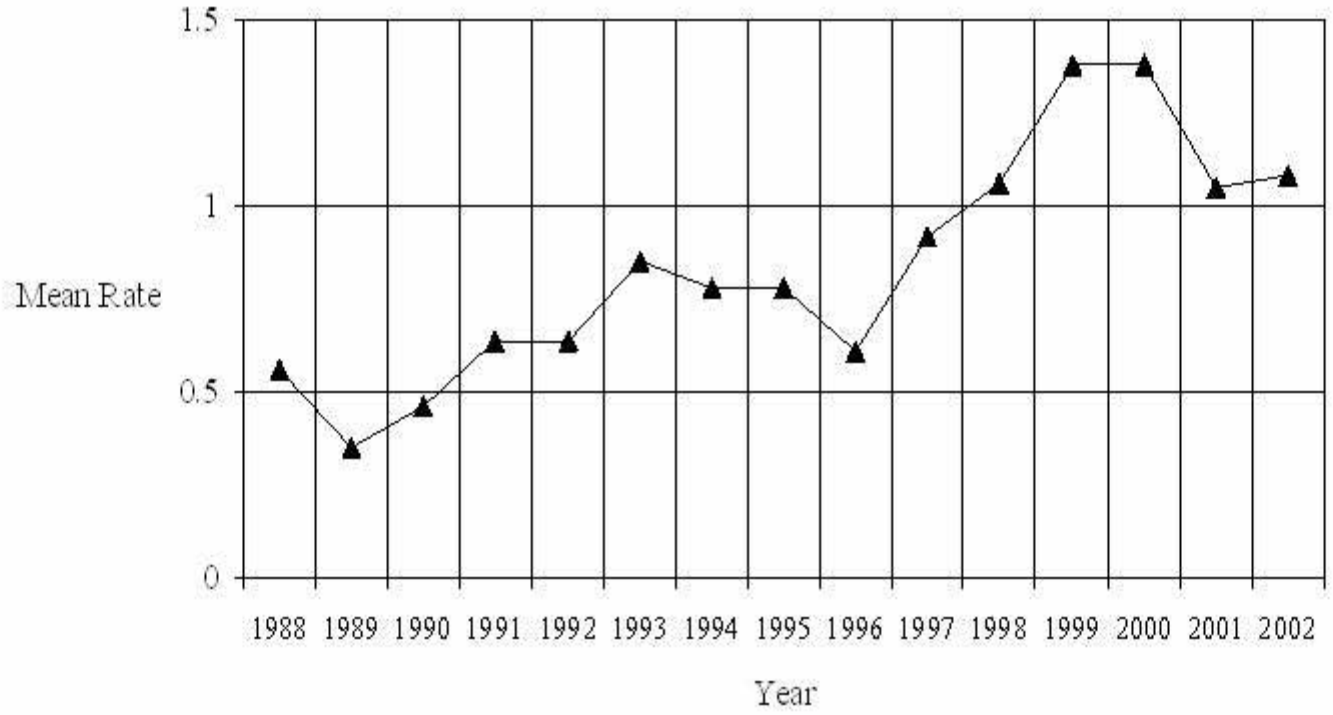
N=19, Adjusted R^2=.238, Sig. F=.020.

Table 5. Mean Benzene Reporting Facilities regressed on AML Rate for white males

Variable	B	Standard Error	Beta	T	Significance
Constant	3.722	.305		12.207	.000
Facilities Mean	.207	.089	.557	2.324	.038

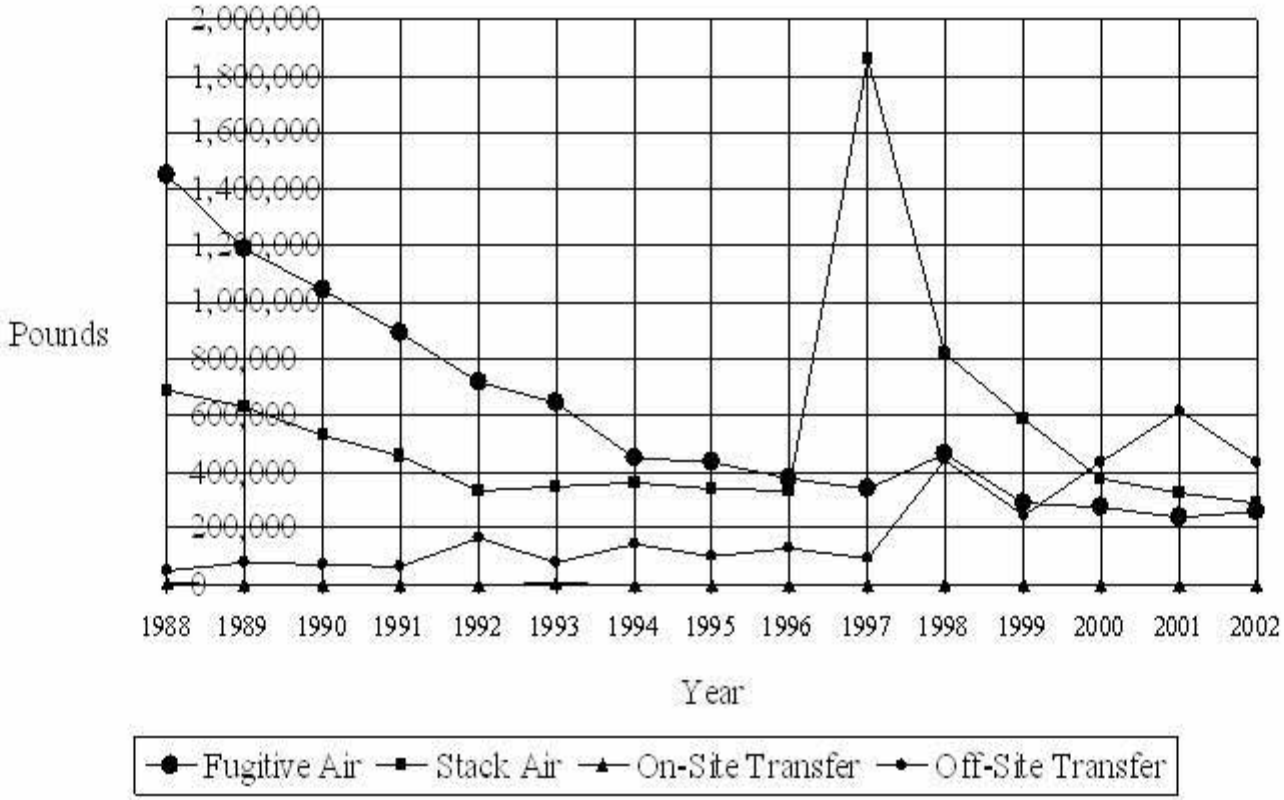
N=14, Adjusted R^2=.253, Sig. F=.038.

Figure 1. Acute Myelogenous Leukemia in Louisiana, 1988-2002.



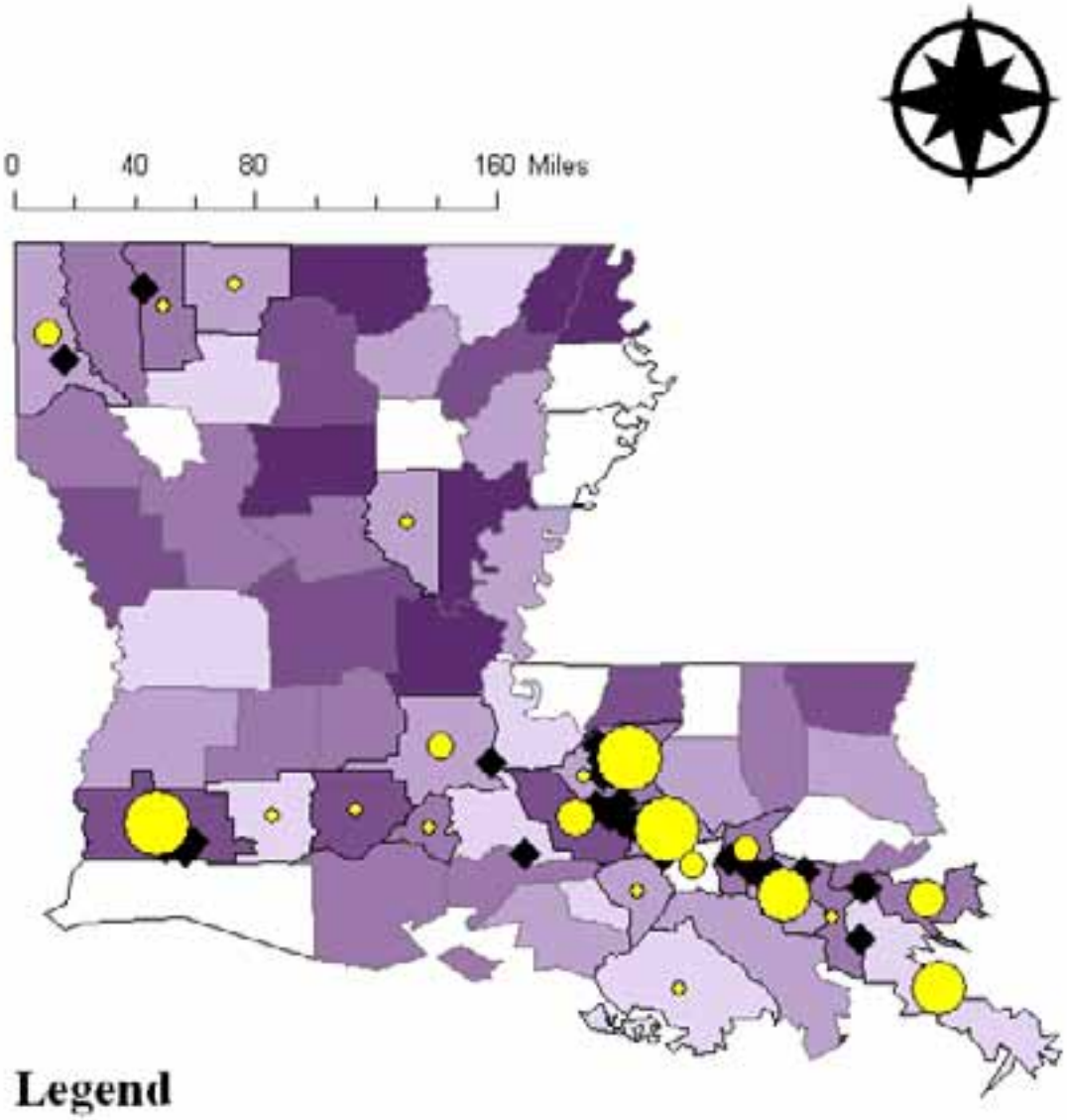
Source: Louisiana Tumor Registry, Louisiana State University, Health Sciences Center.

Figure 2. Benzene Releases in Louisiana, 1988-2002.



Source: Right-to-Know Network. (2006). Toxic Release Inventory. Washington, DC: OMB Watch.

Acute Myelogenous Leukemia Rates, Benzene Reporting Facilities, and Chemical Releases in Louisiana, 1988-2002.



Legend

Acute Myelogenous Leukemia

- 1.5 - 2.5 Cases per 100,000
- 2.6 - 3.1 Cases per 100,000
- 3.2 - 3.8 Cases per 100,000
- 3.9 - 4.9 Cases per 100,000
- 5.0 - 5.8 Cases per 100,000

Benzene Reporting Facilities 2002

Total Releases of Benzene

- 05 - 174007 Pounds
- 174808 - 992534 Pounds
- 992535 - 2613248 Pounds
- 2613249 - 3738270 Pounds
- 3739271 - 4121259 Pounds

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Primary Author's Name: Ronald A. Harris
Organization: Southern University
Address: P.O. Box 96560
Baton Rouge, LA 70813
Title of Paper: Environmental Pollution...
Telephone: (225) 771-2034
Fax Number: (225) 771-3105
E-Mail Address: rwow@yahoo.com
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